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ARTICLE

Epidemiology of *Acinetobacter* spp.-associated healthcare infections and colonization among children at a tertiary-care hospital in Saud Arabia: a 6-year retrospective cohort study

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Abstract A retrospective cohort study was conducted among hospitalized children less than 12 years of age who had *Acinetobacter* spp. isolated from ≥1 cultures between October 2001 and December 2007 at King Abdulaziz Medical City in Riyadh, Saudi Arabia. Children with multidrug-resistant (MDR) *Acinetobacter* spp. healthcare-associated infections (HAIs) were compared to children with antimicrobial-susceptible *Acinetobacter* spp. HAIs and to children colonized with *Acinetobacter*. Children with MDR *Acinetobacter* spp. HAIs were older ($p=0.01$), more likely to be admitted to an intensive care unit (ICU) ($p=0.06$), and had a higher mortality rate ($p=0.02$) than colonized children. Children with MDR *Acinetobacter* spp. HAIs were older than children with antimicrobial-susceptible *Acinetobacter* spp. HAIs ($p=0.0004$), but their mortality rates were similar. Among children with MDR *Acinetobacter* spp. HAIs, burn injuries were the most common underlying illness. HAIs caused by MDR or susceptible *Acinetobacter* spp. occurred after prolonged hospitalization, suggesting nosocomial acquisition. Patients infected with MDR *Acinetobacter* spp. frequently received inappropriate empiric therapy (73.9 %). Further studies are needed in order to identify effective strategies to prevent nosocomial transmission and effective ways of improving patient outcomes.

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Introduction

Acinetobacter spp. survive for prolonged periods in the healthcare environment and on the hands of healthcare workers [1–4]. These organisms are easily transmitted in hospitals and can cause serious healthcare-associated infections (HAIs), particularly among ill children and neonates. These organisms also cause outbreaks that can be difficult to control. Moreover, *Acinetobacter* can acquire numerous resistance genes, becoming resistant to most antimicrobial agents, thereby, seriously complicating empirical therapy

for HAIs among critically ill patients in units where this organism is endemic or epidemic [3, 5, 6].

Hu and Robinson recently published a systematic review of *Acinetobacter* infections in children. They noted that most cases occurred on pediatric wards and intensive care units (ICUs), while outbreaks occurred mainly in neonatal ICUs [2]. *Acinetobacter* can be transmitted to other patients by contaminated equipment or by healthcare workers' hands [7]. In addition, several studies found that *Acinetobacter* colonization may precede infections in certain patients [8–10].

The objective of this study was to describe the epidemiology of *Acinetobacter* spp. HAIs and of colonization among hospitalized children in a tertiary-care hospital in Saudi Arabia. We also sought to assess the outcomes of hospitalized children with HAIs or colonization caused by *Acinetobacter* spp.

Methods

Study site and period

King Abdulaziz Medical City in Riyadh (KAMC-R) is a 900-bed tertiary-care hospital that serves National Guard employees and their dependants. KAMC-R has 90 pediatric inpatient beds, 121 intensive care beds, including three pediatric ICUs (medical/surgical, neonatal, and cardiac), and three step-down units, of all which care for pediatric patients and a shared burn unit with adult patients.

Definitions

The National Healthcare Safety Network's (NHSN) definitions were used to identify HAIs [11]. An *Acinetobacter* spp. isolate was considered to be multidrug-resistant (MDR) if it was resistant to three or more of the commonly prescribed antimicrobial classes, including third- and fourth-generation cephalosporins, broad-spectrum penicillins, aminoglycosides, quinolones, and carbapenems. Isolates resistant to all classes except colistin and tigecycline were considered to be pan-resistant. For simplicity, pan-resistant and MDR isolates were both designated as MDR in this manuscript.

Antimicrobial treatment was considered to be empirical when agents were initiated before in vitro susceptibility testing results were available. Antimicrobial therapy was considered to be appropriate if the *Acinetobacter* spp. isolate was susceptible to at least one of the agents administered empirically.

Study design and participants

The microbiology database of KAMC-R was searched to identify all records for children less than 12 years old with cultures that grew *Acinetobacter* spp. between October 2001 and December 2007. Patients who had community-acquired infections (CAIs) or who were outpatients when the cultures were obtained were excluded from the study. Patients who had MDR *Acinetobacter* spp. isolated from one or more cultures and who met NHSN definitions for HAIs were included in the "MDR infection group." Patients who had antimicrobial-susceptible *Acinetobacter* spp. isolated from one or more cultures and who met NHSN definitions for HAIs were included in the "susceptible infection group" (i.e., the first comparator group). Patients who had either a susceptible or MDR *Acinetobacter* spp. isolated from one or more cultures and who did not meet NHSN definitions for HAIs and did not have a CAI were included in the "colonized group" (i.e., the second comparator group). For each patient, the index date was the date of the first culture that grew *Acinetobacter* spp.

The following clinical data were abstracted from the patients' medical records: demographics, admission diagnosis, underlying chronic or inherited diseases, co-morbidities, total parental nutrition (TPN), dialysis, antimicrobial therapy at the time cultures were obtained, changes in the antimicrobial therapy after the culture and susceptibility data were available, the number of hospital days before the index date, the number of ICU days before the index date, the patient's status at discharge, and the date and cause of death, when applicable. The following outcome measures were evaluated for each patient: total length of hospital stay, length of hospital stay after the index date, ICU admission (yes or no), length of ICU stay after the index date if applicable, and in-hospital mortality within the first 14 days after the index date.

Antimicrobial susceptibility testing

Gram-negative organisms were identified and tested for susceptibility, and the results were interpreted according to the guidelines of the Clinical and Laboratory Standards Institute (CLSI). *Acinetobacter* spp. were identified using an automated system (MicroScan WalkAway, Siemens®) and/or API 20E if MicroScan did not identify the pathogen. Only one isolate per patient was included in the current analysis.

Antimicrobial susceptibility testing was performed by either the breakpoint method, using an automated system (MicroScan WalkAway, Siemens) or by the E-test minimum inhibitory concentration method (AB Biodisk), using E-test strips on Mueller–Hinton agar plates. The isolates were tested to evaluate their susceptibility to the following

antimicrobial agents: amikacin, gentamicin, meropenem, imipenem, piperacillin–tazobactam, cefepime, ceftazidime, and ciprofloxacin. Colistin and tigecycline susceptibility were performed only for MDR isolates and when the patient needed treatment with either of these antimicrobial agents. Quality control was performed by testing these same antimicrobials against *Escherichia coli* ATCC 25922, *E. coli* ATCC 35218, *Pseudomonas aeruginosa* ATCC 27853, and *Enterococcus faecalis* ATCC 29212 to check the thymidine level on Mueller–Hinton agar plates.

Statistical analysis

Descriptive analyses were done by calculating frequencies and percentages for categorical variables and by calculating means and standard deviations for continuous variables. Bivariable analyses were done using the Chi-squared test or the *t*-test, as appropriate. We carried out stepwise multivariate logistic regression analyses to assess the association between HAIs caused by either MDR or susceptible *Acinetobacter* spp. and in-hospital mortality, controlling for age, gender, co-morbid diseases, and ICU admission. Odds ratios (ORs) and 95 % confidence intervals (95 % CIs) were calculated for categorical variables. A *p*-value of less than 0.05 was considered to be statistically significant. Data management and analyses were performed using Statistical Analysis Software (SAS) [12].

Results

A total of 175 patients less than 12 years of age had 177 episodes during which *Acinetobacter* spp. was isolated from clinical cultures. Medical records were not available for 55 patients. Only the first episode was included in the study for the two patients who each had two *Acinetobacter* spp. infections. Medical records for 120 (67.8 %) unique patients were reviewed.

Thirty-seven patients, who did not meet the criteria for an HAI, comprised the *Acinetobacter* colonized group. Of the 68 infected patients, 23 (33.8 %) met the criteria for an HAI with an MDR *Acinetobacter* (MDR infection group) and 45 (66.2 %) patients met the criteria for an HAI with an antimicrobial-susceptible *Acinetobacter* (susceptible infection group; Table 1).

The number of patients in each of the three groups tended to increase over the study period. The differences between the three groups at each time point did not reach statistical significance (Fig. 1; $p=0.38$). We did not assess the significance of the trends because we did not know the denominators.

Table 1 Demographics of children with multidrug-resistant (MDR) *Acinetobacter* spp. healthcare-associated infections (HAIs), children with antimicrobial-susceptible *Acinetobacter* spp. HAIs, and children colonized with *Acinetobacter* spp. at King Abdulaziz Medical City, Riyadh, Saudi Arabia, October 2001–December 2007

Variable	MDR *Acinetobacter* infections ($n=23$)	Antibiotic antimicrobial-susceptible *Acinetobacter* spp. infections ($n=45$)	*p*-value for MDR vs. antibiotic-susceptible infections	OR (95 % CI)	*Acinetobacter* spp. colonized patients ($n=37$)	*p*-value of MDR infections vs. colonization	OR (95 % CI)
Mean age, years (SD)	5.3 (4.0)	1.8 (3.5)	0.0004	1.25 (1.09–1.43)	2.7 (3.6)	0.01	1.19 (1.03–1.37)
Female	12 (52.2 %)	23 (51.1 %)	0.93	1.04 (0.38–2.85)	14 (37.8 %)	0.28	1.79 (0.62–5.14)
Cardiac disease	3 (13.0 %)	23 (51.1 %)	0.002	0.14 (0.04–0.55)	16 (43.2 %)	0.01	0.19 (0.05–0.78)
Burn	12 (52.2 %)	1 (2.2 %)	2×10^{-6}	48.0 (5.62–409.73)	4 (10.8 %)	6.6×110^{-4}	9.0 (2.40–33.74)

MDR = multidrug-resistant; OR = odds ratio; CI = confidence interval; SD = standard deviation

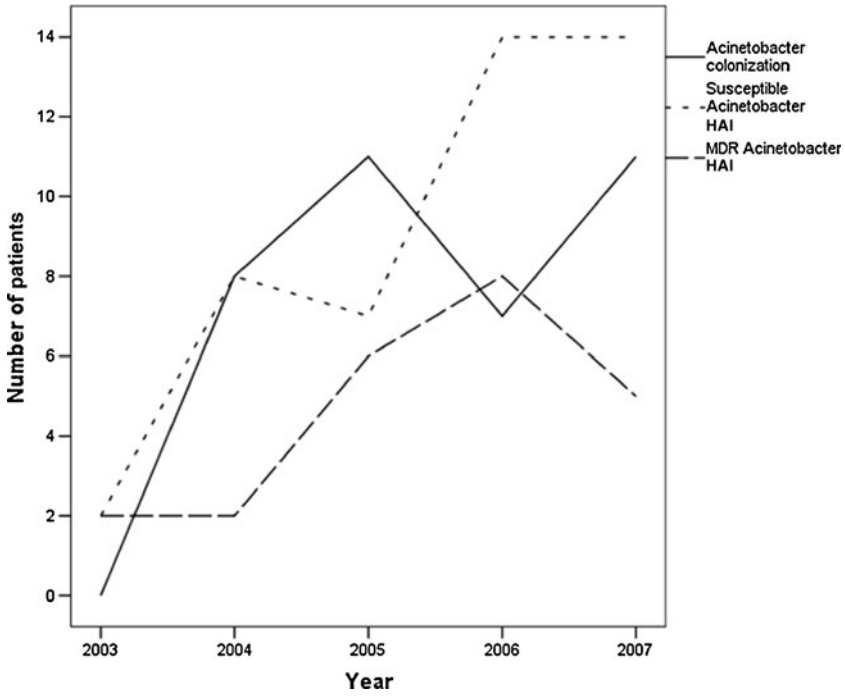

Fig. 1 Number of patients below the age of 12 years with cultures that grew *Acinetobacter* spp. who were hospitalized at King Abdulaziz Medical City, Riyadh, Saudi Arabia, October 2001–December 2007

Children with MDR *Acinetobacter* spp. HAIs compared to children with susceptible *Acinetobacter* spp. HAIs and children colonized with *Acinetobacter* spp.

Children with MDR *Acinetobacter* spp. HAIs were significantly older than children with susceptible *Acinetobacter* spp. HAIs and children colonized with *Acinetobacter* spp. (Table 1). Among children with MDR *Acinetobacter* spp., burns were the most common illness (Table 1). Ventilator-associated pneumonia (VAP) was the most common HAI caused by MDR *Acinetobacter* spp. (Table 2).

The total length of hospital stay (LOHS) and the LOHS after the index date were similar in all three groups (Table 3). The LOHS before the index date was longer for those with susceptible *Acinetobacter* spp. HAIs compared with those with MDR *Acinetobacter* spp. HAIs [p=0.02; OR=0.98 (0.95–1.00)]. The majority of infections with either MDR (65.2 %) or susceptible (62.2 %) *Acinetobacter* spp. HAIs were acquired in an ICU.

Treatment and outcome

Treatment with inappropriate antimicrobial therapy was significantly more frequent for patients with MDR *Acinetobacter* spp. HAIs than for patients with susceptible *Acinetobacter* spp. HAIs. Among patients with susceptible *Acinetobacter* spp.

Table 2 Types of infections among children with MDR *Acinetobacter* spp. HAIs and among children with susceptible *Acinetobacter* spp. HAIs at King Abdulaziz Medical City, Riyadh, Saudi Arabia, October 2001–December 2007

Type of infection	MDR *Acinetobacter* spp. HAIs, *n* (%)	Antimicrobial-susceptible *Acinetobacter* spp. HAIs, *n* (%)
Ventilator-associated pneumonia	8 (34.8 %)	12 (26.7 %)
Central line-associated bloodstream infection	6 (26.1 %)	12 (26.7 %)
Bloodstream infection not associated with central lines	3 (13.0 %)	12 (26.7 %)
Catheter-associated urinary tract infection	2 (8.7 %)	1 (2.2 %)
Burn wound infection	2 (8.7 %)	1 (2.2 %)
Central nervous system infection	0 (0.0 %)	1 (2.2 %)
Peritonitis	0 (0.0 %)	3 (6.7 %)
Soft tissue infection	1 (4.4 %)	1 (2.2 %)
Surgical site infection	0 (0.0 %)	1 (2.2 %)
Conjunctivitis	0 (0.0 %)	1 (2.2 %)

MDR = multidrug-resistant; HAIs = healthcare-associated infections

Table 3 Unadjusted outcomes for children with MDR *Acinetobacter* spp. HAIs, children with antimicrobial-susceptible *Acinetobacter* spp. HAIs, and children colonized with *Acinetobacter* spp. at King Abdulaziz Medical City, Riyadh, Saudi Arabia, October 2001–December 2007

Variable		MDR *Acinetobacter* spp. infections (n=23)	Antibiotic-susceptible *Acinetobacter* spp. infections (n=45)	p-value of MDR vs. antibiotic-susceptible infections	OR (95 % CI)	*Acinetobacter* spp. colonization (n=37)	p-value of MDR infections vs. colonization	OR (95 % CI)
Total LOHS	Mean (SD)	52.4 (34.6)	58.5 (50.5)	0.61	0.99 (0.98–1.00)	49.8 (56.5)	0.84	1.00 (0.99–1.01)
LOHS before index date	Mean (SD)	13.3 (13.8)	30.6 (46.6)	0.02	0.98 (0.95–1.00)	22.3 (56.6)	0.36	0.99 (0.97–1.01)
LOHS after index date	Mean (SD)	39.2 (31.6)	36.7 (44.0)	0.81	1.00 (0.99–1.01)	37.9 (52.7)	0.92	1.00 (0.99–1.01)
ICU admission	Yes	21 (91.3 %)	34 (75.6 %)	0.12	3.40 (0.78–18.80)	26 (70.3 %)	0.06	4.44 (0.89–22.28)
ICU LOS	Mean (SD)	32.9 (30.4)	37.4 (52.0)	0.72	0.99 (0.98–1.01)	32.0 (31.0)	0.92	1.00 (0.98–1.02)
ICU LOS before index date	Mean (SD)	10.3 (10)	8.8 (44.6)	0.86	1.00 (0.99–1.02)	28.8 (66.7)	0.2	0.97 (0.93–1.01)
ICU LOS after index date	Mean (SD)	25.5 (28.3)	29.1 (89.5)	0.84	1.00 (0.99–1.01)	2.8 (59.0)	0.1	1.02 (0.99–1.05)
Infection acquired in ICU	Yes	15 (65.2 %)	28 (62.2 %)	0.81	1.14 (0.40–3.25)	16 (43.20 %)	0.1	2.46 (0.84–7.22)
Mortality for infections acquired in ICU	Yes	7 (46.7 %)	8 (28.6 %)	0.23	2.19 (0.59–8.06)	2 (12.5 %)	0.01	7.88 (1.33–46.62)
Overall mortality	Yes	9 (39.1 %)	12 (26.7 %)	0.29	1.77 (0.61–5.14)	4 (10.8 %)	0.02	5.30 (1.40–20.12)

LOHS = length of hospital stay

MDR = multi drug resistance

HAI = healthcare associated infection

ICU = intensive care unit

HAIs, 15.9 % (7/44) received inappropriate antimicrobial therapy, while 73.9 % (17/23) of patients with MDR *Acinetobacter* spp. HAIs received inappropriate antimicrobial therapy.

A subset analysis among patients with central line-associated bloodstream infection (CLABSI) or bloodstream infection (BSI) caused by either MDR or antibiotic-susceptible *Acinetobacter* spp. revealed that the mortality rate among those receiving inappropriate antimicrobial therapy (42.9 %) was double that for those receiving appropriate antimicrobial therapy (23.1 %), but this difference did not meet statistical significance (p=0.3; data not shown).

The crude mortality rate for children with MDR *Acinetobacter* spp. HAIs was higher than that for children with susceptible *Acinetobacter* spp. HAIs; this difference was not statistically significant (Table 3). The crude (Table 3) and adjusted (Table 4) mortality rates among children with an MDR *Acinetobacter* spp. HAI were higher than those for children colonized with *Acinetobacter*, even when controlling for age, gender, co-morbid diseases, and ICU admission [p=0.003; OR 6.36 (1.89–21.38)]. The crude (Table 3) and adjusted (Table 4) mortality rates for children with susceptible *Acinetobacter* spp. HAIs were higher than those for children colonized with *Acinetobacter*, but these differences were not statistically significant.

Antimicrobial susceptibility results

Greater than 70 % of the isolates of MDR *Acinetobacter* spp. were resistant to aminogylcosides, quinolones, and higher generation cephalosporins, and most isolates were resistant to piperacillin–tazobactam (Table 5).

Discussion

To our knowledge, this is the first study describing the epidemiology of healthcare-associated *Acinetobacter* spp. infections and outcomes of these infections in children during an endemic period [2, 13]. Our study had several important findings. First, patients with MDR *Acinetobacter* HAIs were hospitalized for nearly two weeks and patients with susceptible *Acinetobacter* HAIs were hospitalized for about a month before their infections were identified. This finding is consistent with a nosocomial acquisition. In fact, over 60 % of *Acinetobacter* HAIs in this study were acquired in ICUs, which is consistent with other studies [2, 3, 14, 15]. Similarly, Katragkou et al. found that a prolonged ICU stay was an independent risk factor for acquiring imipenem-resistant *Acinetobacter* spp. among children hospitalized in a pediatric ICU [3]. Previous studies have demonstrated that proper infection control practices can prevent or reduce the transmission of these organisms [16, 17]. For example, Morgan et al. found that healthcare workers caring for patients colonized with MDR *A. baumannii* frequently

Table 4 Results of multivariable logistic regression analyses (controlling for age, gender, co-morbid diseases, and ICU admission) assessing the association of MDR and susceptible *Acinetobacter* spp. HAIs and in-hospital mortality at King Abdulaziz Medical City, Riyadh, Saudi Arabia, October 2001–December 2007

	p-value	OR (95 % CI)
MDR *Acinetobacter* spp. HAIs vs. antibiotic-susceptible *Acinetobacter* spp. HAIs	0.11	2.57 (0.79–8.30)
MDR *Acinetobacter* spp. HAIs vs. *Acinetobacter* spp. colonization	0.003	6.36 (1.89–21.38)
Antibiotic-susceptible *Acinetobacter* spp. HAIs vs. *Acinetobacter* spp. colonization	0.09	3.00 (0.84–10.78)
MDR *Acinetobacter* spp. HAIs or antibiotic-susceptible *Acinetobacter* spp. HAIs vs. *Acinetobacter* spp. colonization	0.03	3.83 (1.16–12.65)

ICU = intensive care unit

MDR = multi drug resistance

HAI = healthcare associated infection

contaminated their gloves, gowns, and hands [7]. Their data suggest that, unless healthcare workers use barrier precautions and hand hygiene appropriately, colonized patients could be a source of *Acinetobacter* spp. for other patients. In a much older study, Corbella et al. showed that reinforcing isolation measures significantly reduced the fecal carriage of MDR *A. baumannii* from 52 % to 31 % ($p<0.01$) and MDR *A. baumannii* infections from 16 % to 11 % (*p*-value not significant) [8].

Second, even though we did not conduct active surveillance for *Acinetobacter* spp. during the study period, we identified a substantial number of colonized pediatric patients where one-third were colonized with MDR *Acinetobacter* spp. Previous studies have shown that colonization with *A. baumannii* precedes clinical infection with this organism in critically ill patients [8–10].

Third, about one-third of the *Acinetobacter* spp. isolates causing HAIs were MDR. This was of particular importance, since children with HAIs caused by an MDR *Acinetobacter* spp. were more likely to receive inappropriate empiric therapy than children with HAIs caused by susceptible isolates. Further analysis also revealed that the mortality among patients with CLABSI or BSI caused by either MDR or antibiotic-susceptible *Acinetobacter* spp. who received inappropriate antimicrobial therapy was double that for those who received appropriate therapy. Although this difference in mortality rates was not statistically significant, we think that the difference is clinically significant. Many studies, mostly among adults, have shown similar high rates of mortality when appropriate therapy is delayed [18, 19]. Appropriate empiric treatment is preferably determined based on local epidemiological data and hospital-generated antibiograms, which is, unfortunately, lacking in most hospitals in developing countries.

Finally, our study had several limitations. First, we retrospectively reviewed microbiology and clinical data. Thus, we could not test isolates further and we could not perform

Table 5 Percentage of *Acinetobacter* spp. isolates resistant to selected antimicrobial agents among isolates causing HAIs or colonizing pediatric patients at King Abdulaziz Medical City, Riyadh, Saudi Arabia, October 2001–December 2007

Antimicrobial agents	MDR *Acinetobacter* spp. isolates, *n* (%)	Antimicrobial-susceptible *Acinetobacter* spp. isolates, *n* (%)	Colonizing *Acinetobacter* isolates, *n* (%)
Amikacin	18 (78.3 %)	0 (0.0 %)	14 (37.8 %)
Gentamicin	21 (91.3 %)	1 (2.3 %)	16 (43.2 %)
Meropenem	17 (77.3 %)	1 (2.3 %)	8 (21.6 %)
Imipenem	14 (60.9 %)	0 (0.0 %)	7 (18.9 %)
Piperacillin–tazobactam	22 (96.7 %)	1 (2.3 %)	13 (35.1 %)
Cefepime	20 (87.0 %)	2 (4.6 %)	13 (35.1 %)
Ceftazidime	18 (78.3 %)	0 (0.0 %)	14 (37.8 %)
Ciprofloxacin	17 (73.9 %)	0 (0.0 %)	11 (29.7 %)
Colistin	0 (0.0 %)	ND	[a]1
Tigecycline	0 (0.0 %)	ND	ND

ND = not done

[a] Unable to calculate percentage

HAI = healthcare associated infection

molecular typing. Thus, we could not determine whether a single clone spread among our pediatric patients, causing an outbreak. Secondly, given our study design, we could not assess risk factors for acquiring *Acinetobacter* infections or colonization, nor were we able to determine whether modifying antimicrobial therapy affected the outcome for patients infected with MDR isolates. Future prospective studies could help us identify effective measures for preventing *Acinetobacter* colonization and infection in pediatric patients and whether outcomes improve if antimicrobial therapy is modified based on the results of susceptibility testing.

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Ethical approval This work was conducted after obtaining IRB approval from King Abdullah International Medical Research Center, King Saud University for Health Sciences, Riyadh, Saudi Arabia. Finally, this work has been partially sponsored by King Abdulaziz City for Science and Technology.

Conflict of interest The authors declare that they have no conflict of interest.

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